

MASSIVE SYSTEMIC AIR EMBOLISM FOLLOWING A BLUNT CHEST INJURY

Dawoud A. El-Halabi, MS, FRCSI; Adel Rizk Botros, MD;
Elizabieta D. Nowacka, MD

Systemic arterial air embolism is a life-threatening problem. It occurs most frequently during open heart surgery,¹ but also in rare cases of left ventriculography,² and penetrating and blunt chest trauma.³ It can also occur as a sequelae to venous air embolism in patients with arteriovenous shunt (paradoxical air embolism). Venous air embolism may occur in any condition where an open vein above the right atrium level is exposed to the atmosphere, e.g., surgical procedures on the head and neck, neurosurgical procedures,⁴ pelvic operations in Trendelenburg position, during gas insufflation in laparoscopy, and during insertion or removal of intravascular catheter, particularly central venous and pulmonary artery catheters.⁵

This paper reports a case of massive systemic air embolism as a result of a blunt chest trauma, which is a rare association.

Case Report

A 25-year-old male patient was involved in a road traffic accident. He arrived at the Surgical Casualty Department conscious and irritable with labored breathing. He was moving all limbs and was bleeding from a cut wound on the scalp and from the oral cavity. His heart rate and blood pressure were 140/min. and 130/90 mm Hg, respectively. Neck movement was free in all directions. Chest auscultation revealed bilateral crepitation with diminished air entry on the right side. The abdomen was soft with no signs of internal injury. There were fractures of both the mandible and the shaft of the left humerus. The patient was started on Ringer lactate solution and hema-cel, and 50 mg of pethedine was given intravenously to relieve pain. In addition, 5 mg Dormicum and 100 mg suxamethonium were given to help the direct-vision laryngoscopy and oral endotracheal intubation. Manual ventilation by Ambubag O₂ (10 L) and Tracrium (40 mg iv), were done during shifting of the patient to the

Intensive Care Unit, where he was subjected to controlled mechanical ventilation with slight low-tidal volume (500 mL) and moderately high respiratory rate (18/min.) without PEEP (positive end-expiratory pressure), to make the peak airway pressure as low as possible. FIO₂ (fraction of inspired oxygen) was 0.6 and oxygen saturation was 94% at the start of the mechanical ventilation. The patient's blood pressure, heart and respiratory rate, temperature, oxygen saturation, ECG, urine output and central nervous pressure (CVP) were continuously monitored.

Chest x-ray revealed intact ribs with a minimal right-side pneumothorax. A right-side chest tube was inserted for underwater seal drainage. X-ray of the skull, cervical spine, pelvis and long bones confirmed the clinical findings. Intravenous catheter was inserted into the right internal jugular vein, the urinary catheter and nasogastric tube also were inserted, and the correct position of both the chest tube and the CVP catheter were checked by chest x-ray. IV fluids (colloids and crystalloids) were given and blood was extracted. A renal dose of dopamine (3 µg/kg/min.) was given. Blood samples were taken for evaluating Hb, serum electrolytes, blood sugar and blood gases. One hour after ICU admission, the patient went into a sudden cardiovascular collapse, with a decrease of oxygen saturation. Rapid fluid infusion (hema-cel and Ringer lactate) was started, plus a vasoconstrictor dose of dopamine (20 µg/kg/min. by syringe pump) for the correction of the hypotension, intermittent administration of 1 mg atropine sulphate for correction of the bradycardia, and pure oxygen ventilation for the correction of hypoxemia. CT scans revealed normal appearance of the brain and cervical spine, severely contused lungs with a laceration of the middle lobe of the right lung, and a rim of air in the right pleural cavity and air filling the left side of the heart, aorta and hepatic vessels (Figures 1, 2 and 3). The cardiothoracic surgeon was called, and after examining the patient found he was beyond salvage. The patient died two hours after his arrival in spite of the resuscitative measures taken.

Discussion

Although systemic air embolism frequently occurs after

From the Departments of Surgery (Dr. El-Halabi), Anesthesia (Dr. Botros), and Radiology (Dr. Nowacka), Al-Adan Hospital, Ministry of Health, Kuwait.

Address reprint requests and correspondence to Dr. El-Halabi: P.O. Box 551619, Rikka 53457, Kuwait.

Accepted for publication 7 June 1998. Received 21 March 1998.

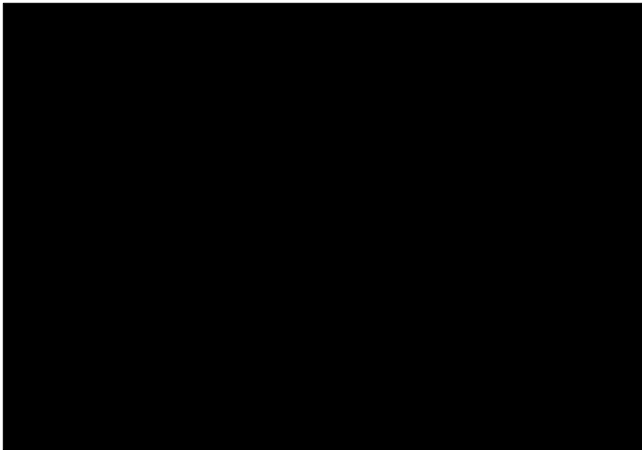


FIGURE 1. Chest CT scan at the level of the heart showing massive contusion of both lungs, laceration in the middle lobe of the right lung and air-fluid level inside the left ventricle and descending aorta.

penetrating thoracic injuries, it can also occur after blunt chest trauma, as we have just reported. Similar findings in three patients following blunt chest trauma have been previously reported.³ Systemic air embolism that occurs in either blunt or penetrating chest trauma is attributed to the associated bronchopulmonary venous fistula.⁶

The outcome from systemic air embolism depends on the amount and the rate of air entrance to the systemic circulation, the organ supplied by the blocked artery, whether it can withstand ischemia for any length of time, the time taken for the diagnosis to be made, and whether facilities exist for adequate management. Massive systemic air embolism is a fatal condition, and clinically should be suspected if a sudden cardiovascular collapse accompanies chest trauma,⁷ beside the appearance of air bubbles in the arterial blood samples. Systemic air embolism is often noticed after the initiation of mechanical ventilation, so anesthesiologists must be aware that the patient with chest trauma is at risk of systemic air embolism during mechanical ventilation, especially with high-peak airway pressure.⁸ The initiation of systemic air embolism in a patient with chest trauma after mechanical ventilation is attributed to bronchopulmonary pressure gradient that favors transmission of air through a traumatic bronchial to pulmonary venous communication, and this has been proved by experimental study on animals.⁸ Early detection of systemic air embolism is vital to allow the possibility of salvaging such patients. The presence of air bubbles in both retinal and coronary arteries and the laterization of focal neurological signs with no head injury are common manifestations of systemic arterial air embolism. Thoracic CT scan and transesophageal echocardiography⁹⁻¹¹ are helpful in early detection of systemic air embolism. Frothy arterial blood means that a large volume of air entered the systemic circulation, and salvage is not possible in nonspecialized centers. Air in the coronary circulation may be one of the major causes of the pump failure, while air in

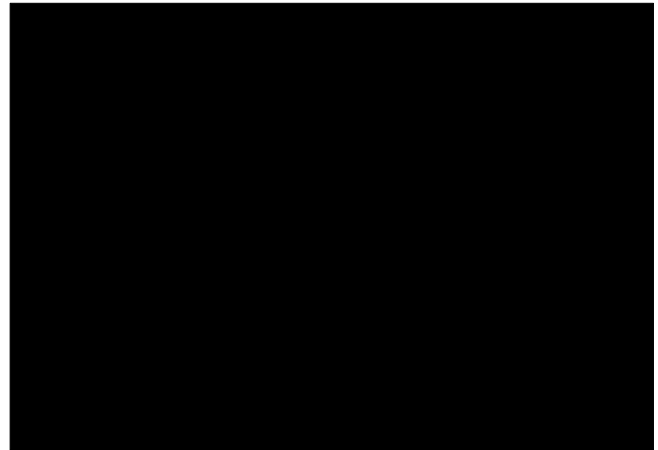


FIGURE 2. Chest CT scan at the level of the main vessels showing air fluid in the ascending aorta.

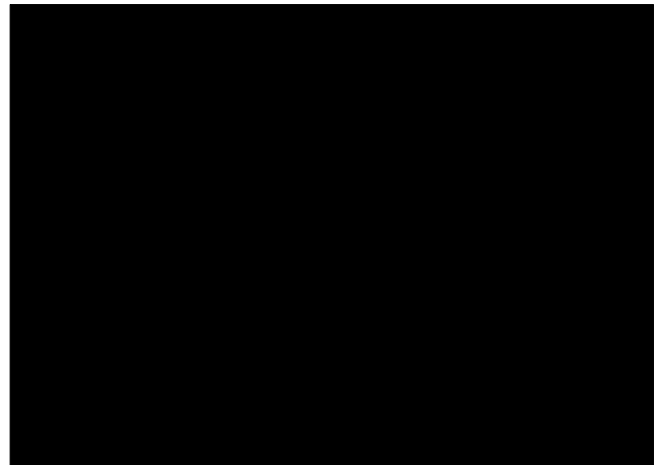


FIGURE 3. Upper abdomen CT scan at the level of the liver showing air in the descending aorta and hepatic artery branches.

the cerebral vascular system may result in profound neurological manifestations.

The cause of death of this reported case was attributed to the irreversible, sudden and severe cardiovascular collapse that occurred as a sequelae to the massive systemic arterial air embolism. In ICU patients, there is a possibility of iatrogenic introduction of air embolism via arterial line if precautions are not taken, but in this reported case this route was excluded because an intra-arterial line was not inserted.

Systemic arterial air embolism that follows chest trauma must be treated as early as possible. Treatment consists of immediate thoracotomy on the injured side and hilar cross-clamping of the lung, and pulmonary vasculature or interruption of ventilation of the involved lung are lifesaving.¹² As prophylactic measures against the occurrence of air embolism after mechanical ventilation in a previously stable patient who has a thoracic injury and is waiting for thoracotomy, it is necessary to avoid excessive

positive pressure ventilation and PEEP during resuscitation via the use of low-tidal volume with high ventilatory rate and the use of high-frequency jet ventilation if available. It is necessary in these circumstances not only to reduce the chance and the severity of air embolism, but also in the long term to decrease the size of air leak and improve the pulmonary healing process.¹³ Expansion of the intravascular space, administration of 100% oxygen to favor the evacuation of air from the closed space and the usage of hyperbaric oxygen are also helpful.¹ Nitrous oxide is contraindicated if air embolism occurs, since it expands the gas collection by entering at a rate exceeding the exit rate of nitrogen. Needle venting of air from the heart by the surgeon, manual cardiac massage, and pressor therapy to increase coronary perfusion pressure beside right decubitus position are standard cardiopulmonary resuscitative measures for the victims of left heart air embolism.

Massive systemic air embolism that may follow chest trauma is a life-threatening problem and needs immediate resuscitation and management in a specialized center.

References

1. Tascano M, Chiavarelli R, Ruvolo G, Macchiarelli A, Scibilia G, Marino B. Management of air embolism during open heart surgery with retrograde perfusion of cerebral vessels and hyperbaric oxygenation. *Thorac Cardiovasc Surg* 1983;31:18-24.
2. Goldenberg I, Shupak A, Shoshani O, Boulos M. Leftventri-culography complicated by cerebral air embolism. *Israel Naval Medical Institute, IDF Medical Corps, Haifa. Cathet Cardiovasc Diagn (US)* 1995;35:331-4.
3. Sauda R, Goarin JP, Rouby JJ, Jacquens Y, Guesda R, Viars P. Systemic gas embolism complicating pulmonary contusion: diagnosis and management using transesophageal echocardiography. *Am J Respir Circul Care Med* 1995;152:812-5.
4. Sakamoto T, Kawaguchi M, Furuya H, Ohnishi H, Karasawa J. Preoperative evaluation for risk of venous air embolism in the sitting position. *J Neurosurg Anesthesiol* 1995;7:124-6.
5. Sing RF, Steffe TJ, Branas CC. Fatal venous air embolism after removal of CVP line. *J Am Osteopath Assoc* 1995;95:204-5.
6. Meir GH, Wood WJ, Symbas PN. Systemic air embolism from penetrating lung injury. *Ann Thoracic Surg* 1979;27:161.
7. Turky D. Initial treatment of patient with extensive trauma. *N Engl J Med* 1991;324:1259-63.
8. Graham JM, Beal AC, Mattox KL, Vaughn GD. Systemic air embolism following penetrating trauma to the lung. *Chest* 1979;72:449.
9. Oka Y, Inouo T, Hong Y, Sisto DA, Storm JA, Frater RW. Retained intra-cardiac air: transoesophageal echocardiogram fordetection of venous air embolism. *J. Anaesthet* 1995;75:447-50.
10. Furuya H, Suzuki T, Okumura F, Kishi Y, Uefuji T. Detection of air embolism by transoesophageal echocardiography. *Anesthesiology* 1983;58:124-9.
11. Tingleff J, Joyce FS, Pettersson G. Intraoperative echocardiography study of air embolism during cardiac operation. *Ann Thorac Surg* 1995;60:673-7.
12. Hanowell LH. Perioperative management of thoracoabdominal trauma. In: Grande CM, editor. *Textbook of Trauma, Anesthesia and Critical Care*. St Louis, Missouri: Mobsy Year Book Inc., 1993:559-82.
13. Shackdard SR. Blunt chest trauma, the intensivist's perspective. *J Intensive Care Med* 1986;1:125.